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<input type="checkbox"/>	L4	L2 and Actinobacillus	55
<input type="checkbox"/>	L3	L2 Actinobacillus	2772
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☐ 2. [20040202678](#). 10 Mar 04. 14 Oct 04. Actinobacillus pleuropneumoniae subunit vaccine. Segers, Ruud Philip Antoon Maria. 424/200.1; 435/252.3 435/471 536/23.7 A61K039/02 C07H021/04 C12N009/14 C12N001/21 C12N015/74.

☐ 3. [6783764](#). 31 Aug 99; 31 Aug 04. Actinobacillus pleuropneumoniae subunit vaccine. Segers; Ruud Philip Antoon Maria, et al. 424/236.1; 424/184.1 424/192.1 424/193.1 424/197.11 424/234.1 424/278.1 424/9.2 435/220 435/235.1 435/340 930/200. A61K049/00 A61K039/00 A61K039/38 A61K039/385 A61K039/02 .

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Mol Microbiol. 1994 Oct;14(2):207-16:

PMID: 7830567 [PubMed - indexed for MEDLINE]

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Structural analysis of the *Actinobacillus pleuropneumoniae*-RTX-toxin I (ApxI) operon.

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Unidad de Microbiología e Inmunología, Facultad de Veterinaria, Universidad de León, Spain.

The involvement of the RTX haemolysins (ApxI and ApxII) of the swine pathogen *Actinobacillus pleuropneumoniae* in virulence was investigated using haemolysin-deficient mutants constructed by a mini-Tn10 mutagenesis procedure. Two types of haemolysin mutant with single insertions of the transposon were obtained from a serotype 1 strain producing both ApxI and ApxII. One presented a complete loss of haemolytic activity because of the absence of ApxI and ApxII production. The other displayed weaker haemolysis than the wild type and produced only ApxII. The chromosomal regions flanking mini-Tn10 were cloned and sequenced. In the non-haemolytic mutant, the transposon had inserted in *apxIB*, a gene involved in the exportation of ApxI and ApxII toxins. The weakly haemolytic mutant resulted from the disruption of the structural gene for ApxI. Both mutations in the *apxI* operon were associated with a significant loss of virulence for mice and pigs, demonstrating that haemolysins are involved in *A. pleuropneumoniae* pathogenicity. The non-haemolytic mutant was apathogenic and the weakly haemolytic mutant retained some virulence for pigs, suggesting that both ApxI and ApxII are needed for full virulence.

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